Amendments to the Specification

Amend the paragraph beginning on page 12, line 27, and ending on page 13, line 7, as follows:

A non-exhaustive list of disorders in which T cells are involved/recruited include:

- Allergic diathesis e.g. Delayed type hypersensitivity, contact dermatitis
- Autoimmune disease e.g. SLE, rheumatoid arthritis, multiple sclerosis, diabetes, Guillain-Barre syndrome, ~~Hashimotos disease~~ Hashimoto's disease, pernicious anaemia
- Gastroenterological conditions e.g. Inflammatory bowel disease, ~~Chrons disease~~ Chron's disease, primary biliary cirrhosis, chronic active hepatitis
- Skin problems e.g. psoriasis, pemphigus vulgaris
- Infective disease e.g. AIDS virus, herpes simplex/zoster
- Respiratory conditions e.g. allergic alveolitis,
- Cardiovascular problems e.g. autoimmune pericarditis
- Organ transplantation
- Inflammatory conditions e.g. myositis, ankylosing spondylitis
- Any disorder where T cells are involved/recruited.

Amend the paragraph beginning on page 18, line 34, and ending on page 19, line 12, as follows:

PLNC Experiment 2. In this experiment, the background counts in wells with no antigen were very high, above 10000 cpm (Figure 3). Even so, the vehicle control was much higher at 40000cpm, so the results were still interpretable. The aims of this ~~expriment~~ experiment were to use the more robust model of PLNC cultures to again test peptides alone and in combination. As different peptides would be hypothesised to work on the different parts of the T-cell receptor from which they were derived (Table 2), peptides from different chains used in combination might act synergistically. It can be seen from Figure 3 that core peptide reduced antigen-stimulated T-cell proliferation, whether freshly dissolved or stored for more than three months at 4°C. Peptide P also showed activity. Peptides M and N did not reduce proliferation. Combinations of peptides M+CP, CP+P, CP+P+N and P+N+M resulted in reduced 3H-thymidine approximately equal to the average of their individual effects and no synergistic actions of combined peptides was noted.

Amend the paragraph on page 20, lines 10-33, as follows:

The solubility of the peptides ~~were~~ was variable. At the concentration of the stock solutions, 1 mg/ml or 1mM, most peptide solutions looked clear. Exceptions were peptides H, I, O, P, which were turbid or had undissolved particles. Therefore, the true concentration of peptides in solution in the culture wells would be less than those nominated in the case of these partially soluble peptides. Core peptide could be dissolved at 2mg/ml, but was not completely soluble at 5 mg/ml. When 20µl of these stock solutions were added to the wells, 0.2 mg/ml CP was more inhibitory than 0.1 mg/ml, however, 0.5 mg/ml was less effective, as the peptide precipitated upon addition to the well. The vehicle for the peptides, except M and N, was 0.1% acetic acid which gave 0.01%, i.e., 1.75mM in the wells. The HEPES-buffered medium effectively buffered this acidity, but in addition to the acetate concentration, the medium was effectively reduced in concentration to 90%. This did not adversely affect the antigen-stimulated proliferation of primary lymph node cell cultures (data not shown), but had a marked effect on cultures of T-cell lines, reducing tritiated thymidine incorporation by 50%. In these experiments, effects of peptides could still be determined by comparison with the vehicle control. The 0.05M sodium carbonate buffer, used to dissolve peptides M and N, was not as detrimental to line T-cells as acetic acid. Peptide L was not tested as it was extremely insoluble. Interestingly, the only peptide that reduced T-cell proliferation which was not a CP derivative was peptide P, and it also originated from the TCR alpha chain. Peptides K, M and N, from the beta, delta and gamma chains, were soluble in their respective buffers.